Biologic Effects of Lead in Fish

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ABSTRACT

There is no evidence that lead constitutes a health problem to fish in the United States. But there is very little evidence on which to base any firm conclusion. Very few analytic data have been reported on concentrations of lead in fish in natural or experimental conditions. What data are available suggest that soluble lead is not present in natural waters of the United States in concentrations likely to be toxic to fish. There is no published evidence of any trend toward increased concentrations of soluble lead in natural waters. Much of the man-dispersed lead that is eventually washed into natural waters is probably precipitated owing to the presence of carbonates, hydroxides, and organic ligands in the water and settles to the bottom. There is no evidence that lead precipitated on the bottom of natural waterways is harmful to fish.

Contamination of natural waters by effluent from lead mines was recognized long ago in England. A report of the River Pollution Commission of 1874 (cited by Jones, 1964) described the disappearance of fish from streams fouled by effluent from lead mines and deaths of waterfowl, horses and cattle in the vicinity of the streams.

Probably the first definitive experiments on lead poisoning in fish were carried out in England by Carpenter (1924, 1925, 1926). An explanation was sought for the continued absence of fish in rivers passing through old mining areas. Minnows placed in a river in cages remained normal until heavy rains occurred. The concentration of lead in the river suddenly increased from an immeasureable value to 0.3-0.4 mg/l and the minnows died. It was reasoned that the rain dissolved surface lead deposits and carried them into the river.

Acute Toxicity of Lead to Fish

For purposes of this review, acute toxicity will be defined as effects occurring within a few hours to 2 weeks following exposure to lead. These effects are best described by the "coagulation film anoxia" theory (Ellis, 1937; Westfall, 1945; Jones, 1964). When fish are placed in solutions containing lethal amounts of lead, a film of coagulated mucus appears over the entire body and is particularly prominent over the gills. The insoluble material interferes with the respiratory function of the gills, resulting in acute respiratory distress and death by suffocation. This effect is not peculiar to lead; it can be produced by toxic concentrations of other heavy metal ions including zinc, iron, copper, cadmium, mercury, manganese, cobalt, nickel, silver, gold, and aluminum (Carpenter, 1930; Ellis, 1937; Doudoroff and Katz, 1953; and Jones, 1964). Although this effect has been demon-

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strated for many species of fresh-water fish, I am unaware of a similar effect being demonstrated for salt-water fish. It may be that lead precipitates out in sea water before toxic concentrations are attained. Thomas (1915) could not poison killiefish with lead in sea water because the material precipitated out of solution; in fresh water 3 mg/l lead nitrate was fatal in 12 hr.

Available evidence suggests that the principle biochemical lesion occurs externally to the body. Fish placed in a solution containing insufficient lead to cause death formed a film of coagulated mucus over their bodies with concomitant respiratory distress (Carpenter, 1927). Recovery occurred when the film was shed. Analysis of the film for lead accounted for virtually all of the lead in the original solution. Although no lead could be found in the body of the fish, it should be pointed out that the exposure was of short duration and the method of lead analysis (Aub et al., 1926) was not as accurate or sensitive as methods in use today.

Certain species of fish are considerably more susceptible to the toxic effects of lead than others. Carpenter (1927) observed that the action of lead on goldfish was the same as for trout, sticklebacks, and minnows, but the first-named was more resistant. Jones (1938) stated that goldfish appeared able to tolerate indefinitely 1 mg lead/l in soft tap water, in which 0.1-0.2 mg lead/l proved fatal to sticklebacks. The amount and nature of the gill secretions may explain variations in species susceptibility to lead (Jones, 1938). Goldfish produce a copious gill secretion. When exposed to 10 mg lead/l, goldfish produced so much precipitated mucus that the solution became milky, and sediment collected on the bottom of the vessel. Ellis (1937) suggested that if the concentration of a pollutant such as lead is low enough, or if the source is limited so it acts on the fish for only a short time, the secretion of additional mucus might wash away the precipitated material before serious toxicity to the fish occurred.

It is difficult to define what concentration of lead is acutely toxic to fish; experimental results from different laboratories vary considerably. Variables such as the dissolved oxygen concentration, pH of the solution, volume and number of exchanges of the experimental solution, and duration of exposure are not always controlled (Doudoroff and Katz, 1953). Water temperature is an important factor. A 10-degree C. rise in temperature reduces the survival time by 50% (Carpenter, 1927). Probably one of the most important factors is the degree of water hardness. Lead is readily precipitated out of solution as the carbonate or hydroxide so that hard water tends to decrease the effective concentration of lead. As water hardness increased from 14-53 mg/l (expressed as calcium carbonate) the concentration of lead in solution decreased from 8 to 1.6 mg/l (Jones, 1964). Another important point regarding the presence of calcium is that it appears to antagonize the toxic effects of lead (Jones, 1938). For example, in solutions containing 1 mg lead/l (as lead nitrate) with 0, 5, 10, 20, and 50 mg calcium/l (as calcium nitrate or chloride), survival times averaged, respectively, 1, 3, 6, 7, and 10.5 days. In this experiment, precipitation of lead could not account for the decreasing toxicity with increasing concentrations of calcium. It was concluded that calcium somehow prevents the coagulation of mucus by lead. A similar protective effect of calcium was shown for other metals.

Carpenter (1925, 1927) and Jones (1938) have reported some of the lowest demonstrable concentrations of lead (0.1-0.4 mg/l) toxic to fish. These authors used either distilled or soft tap water in their studies, which would be expected to provide optimal conditions for the toxic effects of lead. However, it has been suggested that other species of fish probably are more sensitive to lead than those studied by Carpenter and Jones (Doudoroff and Katz, 1953) and, therefore, concentrations of 0.1 mg lead/l may not represent minimal concentrations of lead toxic to sensitive fish under conditions most conducive to poisoning.

Chronic Toxicity of Lead to Fish

Few studies are available concerning the chronic toxicity of lead to fish. Anemia has been reported to occur in catfish exposed to

solutions of 50 mg lead/l for periods of 16-183 days (Dawson, 1935). Somewhat similar findings were reported to occur with guppies exposed to 1.24 and 3.12 mg lead/l (total water hardness 80 mg/l) for periods up to 129 days (Crandall and Goodnight, 1963). In addition to blood changes, histologic studies revealed renal changes consisting of a lack of lymphoid tissue and expanded tubular lumens, a lack of mesenteric fat, cellular elements in the myocardium suggestive of degenerative changes and retarded gonadal development. There was no demonstrable consistent alteration of the respiratory epithelium nor evidence of an accumulation of coagulated mucus, but the possibility of some damage to the respiratory system was suggested by the frequent finding of granular debris in the branchial blood vessels. The histopathology as well as growth inhibition and retardation of sexual maturity suggested to the authors that the secondary effects of inanition and/or stress were the most prominent features of chronic lead intoxication in fish. Growth inhibition also was observed in salt-water plaice when exposed to solutions of 4 mg lead/l (Dilling et al., 1926).

I am unaware of studies with fresh-water fish where simultaneous concentrations of lead have been measured in fish and water. Such information would be essential for evaluating the ability of fish to concentrate lead from the surrounding medium. Kehoe et al., (1937) and Harley (1970) have reported concentrations of 0.24 and 0.16 μg/g occuring in fresh-water fish. Wetterberg (1966) reported concentrations as high as 12 μg/g in liver, 5.7 μg/g in gills, and 1.4 μg/g in muscle of fish taken from a lake located near a rich lead mine in Sweden. No report was made of analysis of the lake water for lead.

The permissible concentration for soluble lead in drinking water has been set at 0.05 mg/l by the U.S. Public Health Service (1962). Analyses of over 1500 samples from natural water sources near water-treatment plants over a 5-year period throughout the United States revealed measurable quantities of soluble lead in less than 20% of the samples analyzed. A total of 27 samples were over the acceptable limit (Kopp and Kroner, 1970). The highest value recorded was 0.14 mg/l. The authors pointed out that the total concentration of lead in a body of water would be higher than soluble lead because the presence of carbonate and hydroxyl ions and certain organic ligands tends to effect precipitation of lead as insoluble lead salts. This was borne out in a study measuring the concentration of lead in particles larger and smaller than 0.45 microns at various depths in Lake Hamilton, Arkansas (Nix and Goodwin, 1970). The concentration of particulate lead increased with increasing depth. At 26 m the concentrations of lead were 0.004 and 0.014 mg/l, respectively, in particle sizes less than and greater than 0.45 microns.

There isn't enough information available to state with any degree of certainty whether or not the occurrence of lead in natural waters of the United States constitutes a serious threat to fish or to humans eating the fish. I have been unable to find any reports of fish kills due to lead polluting natural waters in the United States. In addition to areas close to water treatment plants, it would appear highly desirable to analyze both water and fish for lead downstream from industrial operations likely to emit lead. An evaluation of lead concentrations in the water and fish of lakes used heavily by motorboats also would be desirable. This should be compared with lakes not used by motorboats. The amount of lead emitted into the water from an outboard motor burning leaded gasoline (0.7 lead/l) appeared to be related to the size of the motor burning leaded gasoline (0.7 g lead/l) appeared to be related to the size of the motor and the speed of operation (English et al., 1963). A 10-hp engine operated at 1/2-3/4 throttle was shown to emit 0.229 g lead/l of gasoline consumed into the water, whereas a 5.6-hp engine operated at full throttle emitted 0.121 g lead/l of gasoline consumed into the water.

Marine Organisms

There is no question that certain marine organisms can concentrate lead present in

sea water. The normal concentration of lead in sea water is stated to be in the order of 30 µg/l (Water Quality Criteria, 1968). Although concentrations of lead reported to occur in seafood are relatively low, they do indicate considerable concentration from the surrounding medium. For example, Schroeder et al., (1961) report a range of 0.17-2.5 µg/g in seafood, with an average of 0.5 µg/g. Only 1 sample exceeded 0.87 µg/g. Harley (1970) reported a concentration of 0.31 µg/g in shellfish. Pringle et al., (1968) report average wet weight concentrations of 0.47, 0.70, and 0.52 µg/g occurring in eastern oysters, soft shell clams and northern quahaugs, respectively. The remarkable ability of the eastern oyster to concentrate lead was demonstrated by experimentally exposing the oysters to flowing sea water containing concentrations of 0.025, 0.05, 0.1, and 0.2 mg lead/l. After 49 days the total accumulations of lead in the oysters amounted to 17, 35, 75, and 200 µg/g wet weight. Oysters exposed to the 2 lower experimental concentrations of lead appeared normal. Oysters exposed to the 2 higher (0.1 and 0.2 mg lead/l) experimental lead concentrations, however, showed considerable atrophy and diffusion of the gonadal tissue, edema, and the hepatopancreas and mantle edge became less distinct. In view of the insolubility of lead in sea water under usual conditions, it would be of immense interest to know if conditions could arise enabling high concentrations of lead to exist in sea water in a chemical form that could be absorbed by marine organisms to the extent shown in this experimental study.

References Cited

Aub, J.C., Fairhall, L.T., Minot, A.S., and Reznikolf, P., 1926. Lead Poisoning. Medicine Monographs, Vol. 7.

Carpenter, K.E., 1924. A study of the fauna of rivers polluted by lead mining in the Aberystwyth district of Cardiganshire. Ann. App. Biol. 11: 1-23.

Carpenter, K.E., 1925. On the biological factors involved in the destruction of river fisheries by pollution due to lead mining. Ann. App. Biol. 12: 1-13.

Carpenter, K.E., 1926. The lead mine as an active agent in river pollution. Ann. App. Biol. 13: 395-401.

Carpenter, K.E., 1927. The lethal action of soluble metallic salts on fishes. Brit. J. Exp. Biol. 4: 378-390.

Carpenter, K.E., 1930. Further researches on the action of metallic salts on fishes. J. Exp. Zool. 56: 407-422.

Crandall, C.A., and Goodnight, C.J., 1963. The effects of sub-lethal concentrations of several toxicants to common guppy *Lebistes reticulatus*. Trans. Amer. Microscop. Soc. 82: 59-73.

Dawson, A.B., 1935. The hemopoietic response in the catfish, *Ameiurus nebulosus,* to chronic lead poisoning. Biol. Bull. 68: 335-346.

Dilling, W.J., Healey, C.W. and Smith, W.C., 1926. Experiments on the effects of lead on the growth of plaice (*Pleuronectes platessa*). Ann. App. Biol. 13: 168-176.

Doudoroff, P., and Katz, M., 1953. Critical review of literature on the toxicity of industrial wastes and their components to fish II. The metals, as salts. Sewage Ind. Wastes 25: 802-839.

Ellis, M.M., 1937. Detection and measurement of stream pollution. U.S. Bur. Fish Bull. (Bull. No. 22) 48: 365-437.

English, J.N., McDermott, G.N., and Henderson, C., 1963. Pollutional effects of outboard motor exhause – laboratory studies. J. Water Poll. Contr. Fed. 35: 923-931.

Harley, J.H., 1970. Discussion on sources of lead in perennial ryegrass and radishes. Environ. Sci. Technol. 4: 225.

Jones, J.R.E., 1938. The relative toxicity of salts of lead, zinc, and copper to the stickleback (*Gasterosteus aculeatus*) and the effect of calcium on the toxicity of lead and zinc salts. J. Exp. Biol. 15: 394-407.

Jones, J.R.E., 1964. Lead, zinc, and copper. The "coagulation film anoxia" theory. In Fish and River Pollution. Ch. 5. Butterworth Press, London.

Kehoe, R.A., Thamann, F., and Cholak, J., 1937. On the normal absorption and excretion of lead. J. Ind. Hyg. 15: 257-300.

Kopp, J.F., and Kroner, R.C., 1970. Trace metals in waters of the United States. A five-year summary of trace metals in rivers and lakes in the United States (Oct. 1, 1962 – Sept. 30, 1967). U.S. Dept. Interior, Federal Water Pollution Control Administration, Division of Pollution Surveillance, Cincinatti, Ohio.

Nix, J., and Goodwin, T., 1970. The simultaneous extraction of iron, manganese, copper, cobalt, nickel, chromium, lead and zinc from natural water for determination by atomic absorption spectroscopy. Atomic Absorption Newsl. 9: 119-122.

Pringle, B.H., Hissong, D.E., Katz, E.L., and Malawka, S.T., 1968. Trace metal accumulation by estuarine mollusks. J. San. Eng. Div; Proc. Amer. Soc. Civil Eng. 94: 455-475.

Thomas, A., 1915. Effects of certain metallic salts upon fishes. Trans. Amer. Fish. Soc. 44: 120-124.

Westfall, B.A., 1945. Coagulation film anoxia in fishes. Ecology 26: 283-287.

Wetterbert, L., 1966. Acute porphyria and lead poisoning. Lancet 1: 498.

Automotive Emissions Control

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ABSTRACT

Motor vehicles account for 38.9% of the total mass of man-made air pollutants in the U.S., according to a recently published HEW inventory. However, when relative harmfulness of individual pollutants is considered, the motor vehicle contribution is reduced to about 12%. Current model motor vehicles emit approximately 83% less hydrocarbons and 70% less carbon monoxide than precontrolled vehicles. By 1976, vehicles are expected to reduce HC emissions by 98%, CO by 97%, and oxides of nitrogen by 90%.

The California Air Resources Board has shown that motor vehicle emissions of HC and CO in the South Coast Basin have been reduced by 18% and 13%, respectively, from peak values which occurred during the mid-1960's. NO_X emissions continued to increase until 1971, but are now declining. Total nationwide emissions from motor vehicles are also declining, in spite of an increasing car population.

Automotive power plants for the balance of this decade will be highly refined derivatives of today's internal combustion engines. Exhaust gas recirculation, thermal reactors, and catalytic converter systems are being developed to meet future requirements, although both customer acceptance factors and emission control are not fully acceptable at this time. Lead-free fuel will be required to insure satisfactory component life, to minimize harmful effects of combustion chamber deposits, and to significantly reduce the emission of particulate matter. Major variations of the reciprocating internal combustion engine, gas turbines, Rankine cycle engines, and electric propulsion systems are also being actively developed to meet future needs for virtually emission-free vehicles.

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For six years, he was responsible for a staff activity which developed and implemented a broad reliability program to coordinate, unify and up-date the reliability methodology of Ford's 12,000-man engineering organization.

A graduate of University of Illinois in mechanical engineering (BSME, 1947), Mr. Simpson is a member of the Society of Automotive Engineers, the Air Pollution Control Association, the Air Quality Committee of the Automobile Manufacturers Association, and the U.S. Department of Health, Education and Welfare Industrial Task Force for review of Control Techniques Documents. He is author of numerous articles and technical papers on automotive emissions and reliability methods.